THE JOURNAL OF
ABNORMAL PSYCHOLOGY

DECEMBER, 1907–JANUARY, 1908

FURTHER INVESTIGATIONS ON THE GALVANIC PHENOMENON AND RESPIRATION IN NORMAL AND INSANE INDIVIDUALS

(From the Psychiatrical Clinic of the University of Zurich)

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THE changes produced in the electrical resistance of the human body by various causes have been studied for many years but as yet no definite results have been reached. Ch. Féré was the first to report on the changes produced by emotions. In a communication made to the Société de Biologie in 1888 he noted that there was a lessening in the bodily resistance when various sensory stimuli were employed, and also that emotions caused the same decrease. R. Vigouroux had been working on the problem of electrical resistance in the human body with patients from the Salpétrière and had reached the conclusion that the old view that the resistance was due to the epidermis was wrong and that the condition of the superficial circulation was the real cause. He thought that variations in resistance were caused by an increased or decreased superficial circulation. Féré accepted these conclusions and added that "l'étude de la résistance électrique peut trouver une application dans les recherches des psycho-physiologues."

Nothing new was reported for several years. A. Vigouroux in 1890 published a report on the study of elec-

trical resistance in melancholics but added nothing; Tarchanoff, Stricher, Sommer and Veraguth have all published the work of the French investigators. The first real psychological researches with the galvanometer were made by Veraguth who worked with Jung's association experiments and this instrument in 1906. In 1906 work was begun in the Psychiatrical Clinic in Zurich to determine if possible the cause of the electrical resistance of the body and the various changes produced in it in normal and insane individuals by various stimuli. The apparatus used consisted of a circuit containing a single element of low E. M. F., a Deprez-d'Arsonval galvanometer of high sensibility, a shunt for lowering the oscillations of the mirror and two brass plates upon which the test-person places his hands and completes the circuit. The galvanometer reflects a beam of light to a celluloid scale to which is attached a movable slide with a visière, which, pushed by hand, follows the moving mirror-reflection. To the slide is attached a cord leading to a so-called ergograph writer which marks the movements of the slide by means of a pen-point on a kymograph-drum which is fitted with endless paper. For marking time a Jaguet chronograph was used and for the moment of stimulus an ordinary electrical marker.

The problem of the cause of the resistance was first attacked and the results given are those obtained by Jung and Binswanger and as yet are unpublished. The resistance was found to vary greatly in different individuals with different conditions of the palmar epithelium. That the epidermis was the seat of resistance was proven by the fact that when the electrodes were placed under the skin the resistance was enormously decreased. This was done by piercing the skin of each arm with a surgical needle and using the needles as electrodes.[1]

The French investigators were unanimous in ascribing the changes in resistance to changes in the blood supply of a part caused by dilatation and contraction of the vessels, the greater the blood supply the lower the resistance and *vice versa*. That the blood supply was not a chief factor

[1] Experiments by Veraguth and Jung and Binswanger.

was proven by exsanguinating the part in contact with the plates with an Esmarch bandage when it was found that the galvanic phenomenon still existed.

That the changes in resistance are not due to changes in contact, such as pressure on the electrodes is shown by the fact that when the hands are immersed in water which acts as a connection to the electrodes the changes in resistance still occur. Pressure and involuntary movements give an entirely different deflection than that which we are accustomed to obtain as the result of an affective stimulus.

The time which elapsed between a stimulus and the change in resistance as shown by the galvanometer suggested some change in the sympathetic nervous system or in some part controlled by it. The sweat-glands seemed to have more influence than any other part in the reduction of the resistance. If the sweat-glands were stimulated there would be thousands of liquid connections between the electrodes and tissues and the resistance would be much lowered. Experiments were made by placing the electrodes on different parts of the body and it was found that the reduction in resistance was most marked in those places where the sweat-glands were the most numerous. It is well known that sensory stimuli and emotions influence the various organs and glands, heart, lungs, sweat-glands, etc. Heat and cold also influence the phenomenon, heat causing a reduction and cold an increase in the resistance. In view of these facts the action of the sweat-glands seems to be the most plausible explanation of the changes in resistance.

The following experiments were made in the winter and spring of 1907, with a view of determining the effect on the galvanic phenomenon and respiration of a series of simple physical and mental stimuli in a number of normal and insane test-persons. The galvanometric changes were noted by the apparatus described above. The respirations were recorded by means of a Marey pneumograph attached to the thorax and leading by means of a rubber tube to a Marey tambour to which is attached a pen-point which writes on the kymograph drum.

The results of pneumographic experiments of various

authors are very conflicting. Delabarre[1] found that attention to sensory impressions increased the frequency and depth of the respirations. Mosso in his work on the circulation in the brain could come to no satisfactory conclusions. Mentz found that every noticeable acoustic stimulus caused a slowing of the respiration and pulse. Zoneff and Meumann found that high grades of attention cause a very great or total inhibition of respiration, while relatively weaker attention causes generally an increase in the rate and a decrease in the amplitude of the respirations. Total stoppage of respiration was found in sensory attention relatively more frequently than in intellectual. Martius notes great individual differences and comes to the conclusion that there is an affect type which differs from the normal rest and is shown by a slowness of the pulse and respiration.

The experiments of the above authors were all made on a limited number of test persons, usually students. Our experiments with the pneumograph were made usually on uneducated men, attendants in the Asylum, and our stimuli were quite different from those used by the other investigators. It is possible that the great difference in our results may depend in part on these facts.

In our experiments care was taken to have the conditions as nearly equal as possible. It was found that different positions of the body, leaning forwards or backwards, for example, caused a change in the level of the respiratory curves. Slight movements of the body and of the limbs did not influence the curves. The tambour itself can cause changes in the recorded curves. The tambour must contain the same amount of air in every case or the curves will be dissimilar. The curve registered is not an exact one owing to the faults of the instruments. In deep inspirations the rubber covering is rendered tense and when the pressure in the chest changes the elasticity of the rubber causes the respirations to be registered in a different way than they really occur.

It must also be borne in mind that the respiratory curves recorded cannot be regarded as ordinary normal

[1] Remarks of Delabarre, Mosso and Mentz are quoted from Zoneff and Meumann.

respirations but only as an experimental normal. No one can breathe naturally with a recording apparatus on his chest and with his attention more or less directed to it. The release from the tension of the experiment is well seen at the end of the experiment where the respirations become deeper and the level of the curve is changed. The pneumograph could not be used with the women on account of the clothing nor could it be used with many of the insane test persons because of their excitability.

The plethysmograph was not used because the sources of error with it are too numerous. Martius has shown that even when the arm and instrument are encased in plaster of Paris involuntary movements occur which render correct interpretations of the results difficult.

In the galvanic curves many sources of error must be avoided. Chief among these is the deflection caused by a movement of the hands. An increase or decrease in the pressure of the hands upon the electrodes causes an instantaneous change in the position of the reflection of the galvanic mirror. This change is sudden and one can hardly voluntarily produce a change in the position of the reflection which resembles that caused by an affective mental process. The ordinary change of position of the hands is shown by an almost vertical rise or fall of the galvanic curve as shown on the kymograph drum. To avoid as far as possible involuntary changes of position, bags of sand were placed on the hands, thus preventing any but voluntary movements. It was found that quite extensive movements of the body could be made without influencing the galvanometric curve. Deep inspirations, sighs, cause more or less of a rise in the curve. In the same curve a sigh occurring after an affective process seems to cause a more extensive rise than one occurring before. Voluntary long inspirations cause little or no disturbance. It must therefore be assumed that sighs are caused by some affective complex, or that they cause such a complex to come into consciousness, or that they cause an unconscious feeling state.

The test persons consist of physicians, attendants and patients suffering from various mental diseases.

The experiment may be divided into six parts, each part consisting of a separate stimulus or series of stimuli of the same kind, physical or psychical. Before each stimulus or series of stimuli the test-person was told in a general way what was to occur. In many individuals after a short period of waiting for a stimulus there were changes in the respiration and in the galvanic curve due to expectation. These expectation-curves will be discussed later.

The measurements of height are in each case the real, *i.e.*, the vertical height. The respiratory rate is given as so many per centimeter which is a purely comparative measurement. In the quiet periods the average rate per centimeter for ten centimeters at the beginning and end of each period are given.

Part I of the experiment consists of a quiet period of four minutes. The test-person was requested to sit as quietly as possible and was told that no stimulus was to be given. In Part II the stimulus was a leaden weight allowed to fall about three feet onto the floor. In Part III the test-person was requested to speak spontaneously, after a minute or so, a word or short sentence and then remain quiet. Part IV consists of three physical stimuli, a low whistle, a weight dropped onto the floor and a picture, picture post-card, shown to the test-person. Part V consists of four sentences spoken by the investigator. The first two were usually some familiar proverb as, "The pitcher goes to the well until it is broken"; the third and fourth were more critical and referred directly to the test-person or to his habits. In several cases single words, such as "eye" and "face," were given. Part VI is again a quiet period of four minutes. The results of each part will be given and the normal test-persons, fifteen in number, will be first considered.

NORMAL TEST-PERSONS

Part I. The galvanometric curve is usually higher at the beginning than a short time afterwards, due to the feeling of expectation and tension caused by the unaccustomed position and the strange experiment. As a rule the curve shows many irregularities caused by the movements of the

hands and body which the test-person makes in adapting himself to a comfortable position, to expectation, to muscular tension, which factor, however, is not great, and to various feeling complexes. In the course of the quiet period oscillations of the galvanic mirror are seen which cannot be accounted for by any movement of the hands or body, by any respiratory change or by any conscious thought or association. We have, therefore, attributed them to the indefinite *feeling* caused by some complex which remains in the subconscious. Every one has experienced these vague feelings, sad or gay, which come without apparent cause, remain but a short time and are soon forgotten. Such a curve was well shown in the case of a well-educated physician with a good power of self-analysis who could not remember any affective thought which had occurred to him during the period.

The inspirations at the beginning of the quiet period are as a rule deeper and more frequent than at the end. At the beginning they average 2.91 per cm. and at the end 2.79 per cm. The average height of the inspirations at the beginning is 12.41 mm., at the end 12.26 mm. The respiratory curve does not show any great or constant change of level in our cases.

In Part II (stimulus a falling weight) the galvanometric curves show great individual differences. In one case, an attendant who was very nervous and frightened at the experiment, the galvanometric deflection was 54 mm. In another case, also an attendant, but of a very phlegmatic disposition, the deflection was only 4.6 mm. The average deflection for fifteen test-persons was 20.6 mm.

The latent time, *i.e.*, the time from the moment of stimulus to the beginning of the rise of the galvanic curve varies from 1.5 to 5.5 seconds. This time while showing individual variations is usually shorter in the cases which show the greatest galvanic reactions and averages 2.87 sec. The time required for the curve to reach its maximum height corresponds roughly to the height, a curve of 54 mm. requiring 11.5 sec. and one of 10 mm. requiring 2.5 sec. The average time is 6.93 seconds.

The inspirations show individual differences in rate and amplitude and the respiratory rate does not vary as the height of the galvanometric curve, as the following table will show.

Height of galv.	Inspir. before stim.	Rise of galv. curve	Fall of galv. curve
54 mm.	3.5 per cm.	3.86 per cm.	3.92 per cm.
18.8 mm.	3. per cm.	2.72 per cm.	2.5 per cm.
4.6 mm.	3. per cm.	2.5 per cm.	2.3 per cm.

Thus the change in rate for a galvanic curve of 54 mm. is not so great as in the case of a curve of 4.6 mm. Whether the respiration is slowed or quickened during the rise of the galvanic curve seems to depend on the individual. The majority, however, show a slowing during the rise and a quickening during the fall of the galvanic curve.

The average number of inspirations before the stimulus is 3.05 per cm., during the rise of the galvanic curve 3.02 cm., and during the fall 3.09 per cm.

The amplitude of the inspirations does not vary directly with the rate. Before the stimulus the average height of the inspirations is 11.75 mm., during the rise of the galvanic curve 10.73 mm., and during the fall of the curve 11.45 mm.

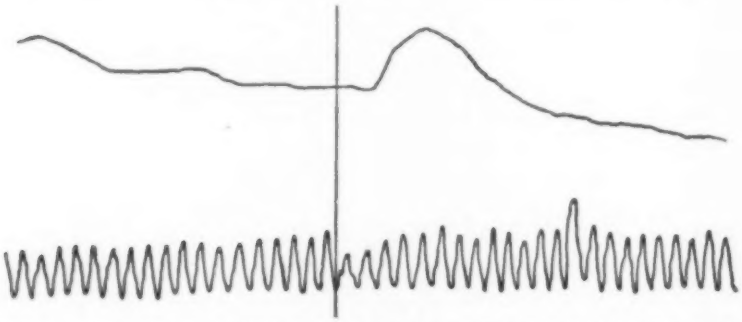

FIG. 1.—Stimulus a falling weight. The resistance was very high at the beginning of the experiment and fell throughout the quiet period and up to the moment of stimulation as shown by the vertical line. The latent time and the lessening in the rate and amplitude of the respirations is well shown.

Part III (spontaneous speaking). In this part the average height of the galvanic curve is less than in the preceding, being 17.9 mm. As a rule the curves of the different test-

persons show little variations in height. Some of the curves show irregularities before the moment of speaking, caused partly by indecision and partly by the preparation for speaking. In the normal test-persons the galvanic curve begins to rise with the moment of speaking or even a little before the moment of speaking.

The number of inspirations per centimeter is decreased during the rise of the galvanic curve and continues to decrease as the curve falls. The average rate before speaking is 3.5 per cm., during the rise of the galvanic curve 3.15 per cm., and during the fall 3.04 per cm. The average height of the inspirations before speaking is 10.08 mm., during the rise of the curve 10.57 mm., and during the fall 11.75 mm. Thus the height increases as the rate decreases.

In Part IV the stimuli are three in number: a falling

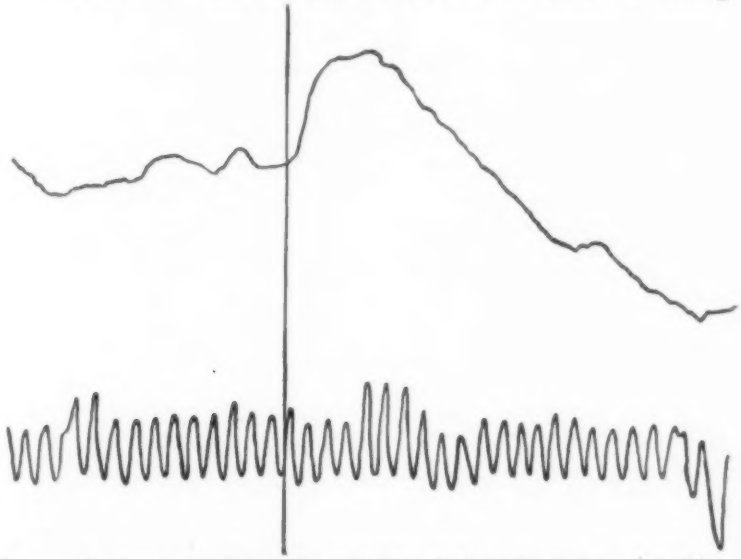

FIG. 2.— Spontaneous speaking. The vertical line indicates the moment of speaking. The irregularities before speaking are well shown in the galvanometric curve. In the respiratory curve the decrease in amplitude during the rise of the galvanometric curve is well shown.

weight, a whistle and a picture. In each case the stimulus is not merely a sensory, visual or auditory one, but has also a psychical component. Practically every stimulus when

perceived or received into consciousness is associated with affective complexes. A low whistle is heard not only as a sound but also as a call and is associated with many past experiences; a picture calls up many other associations. Naturally the personal equation comes into play here to a very great extent.

The results are:

	Weight	Whistle	Picture
Height galv. curve	17.94 mm.	18.2 mm.	19.72 mm.
Latent time	2.55 sec.	2.82 sec.	3.03 sec.
Time to reach max.	6.95 sec.	9.88 sec.	7.47 sec.

In these cases the latent time increases as the height of the galvanometric curve. The time for the curve to reach its maximum varies in the different cases.

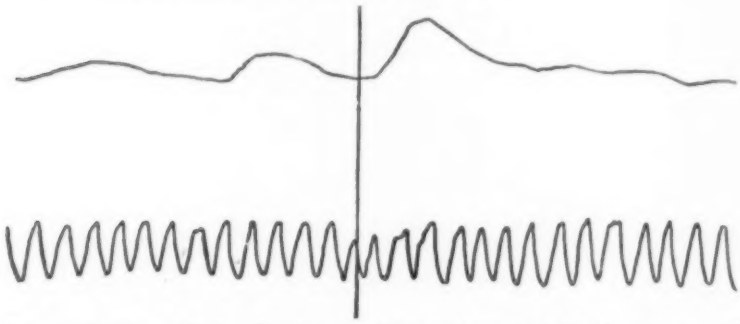

FIG. 3.— Stimulus a whistle. Showing a small expectation curve before the movement of stimulus. The latent period and the changes in the respiratory rate and amplitude are well shown.

The respiratory rate in every case is increased during the rise of the galvanic curve and in one case decreased and in two increased during the fall. The amplitude of the respirations varies in the same way, being less during the rise and increasing in height as the affect passes off. Expressed in tabular form the results are:

	Inspirations per cm.			Height in mm.		
	Weight	Whistle	Picture	Weight	Whistle	Picture
Before stimulus	3.01	2.75	2.88	12.02	12.05	12.46
Rise of curve	3.33	2.77	3.02	10.56	11.35	10.90
Fall of curve	2.76	3.06	3.09	12.32	12.13	11.33

Part V, four short sentences or words were used as stimuli. The sentences were spoken by the investigator and time was allowed between each for the galvanic curve to return to its lowest level. The results are:

	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Height of galv. curve	14.62 mm.	14.48 mm.	19.42 mm.	11.12 mm.
Latent time	3.32 sec.	3.1 sec.	2.83 sec.	3.15 sec.
Time required to reach max.	8.13 sec.	5.82 sec.	7.67 sec.	5.95 sec.

As will be seen in the table the height of the galvanic curve gradually decreases in the second and fourth sentences, while the curve of the third sentence is higher. The gradual decrease in the height of the galvanic curve is to be expected and can be explained by the gradual exhaustion of the affect. The first two sentences were trite ones, and the third was usually one referring to the test-person or one that he could refer to himself, hence the stronger innervation and the increase in the height of the galvanic curve.

The latent time and the time required for the curve to reach its maximum height bear no constant relation to the height of the galvanic curve.

The respiratory curves vary greatly in the different sentences. In two sentences the respiratory rate is decreased and in two increased during the rise of the galvanic curve. The amplitude of the inspirations is always less while the galvanic curve is rising, while the affect is acting, and slowly increases as the affect passes off as the following table will show:

	Inspirations per cm.				Height in mm.			
	1. Sent.	2. Sent.	3. Sent.	4. Sent.	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Bef. stimulus	2.84	2.97	2.71	3.05	12.85	12.59	13.74	12.23
Rise galv. cur.	3.04	2.78	2.57	3.41	11.63	11.27	12.81	11.76
Fall galv. cur.	3.09	2.74	3.13	3.46	12.13	11.98	13.38	13.07

Part VI is a second quiet period of four minutes. As a general rule this part shows fewer irregularities than the first, due to the fact that the test-person had gotten accus-

tomed to the experiment and is comfortably fixed in his place. One marked feature of this part is the change of level of the respiratory curve as soon as the test-person is told that the experiment is ended and he is released from the involuntary tension in which he has been held.

The respiratory rate is slower than in the first quiet period. At the beginning the inspirations are 2.41 per cm., as compared to 2.91 per cm., in the first curve. At the end they are 2.71 per cm. as compared to 2.79 per cm., in the first curve. The height of the inspirations is 12.57 mm., at the beginning as compared to 12.41 mm., in the first curve and 12.17 mm., at the end as compared to 12.26 mm., in the first curve.

What we have designated as expectation curves are changes in the galvanic curve which occur while the test-person is waiting for the stimulus. Naturally they vary according to the individual. Some of our test-persons had absolutely no sign of an expectation curve while others had quite marked ones. These curves are more frequent in the early part of the experiment and are especially marked

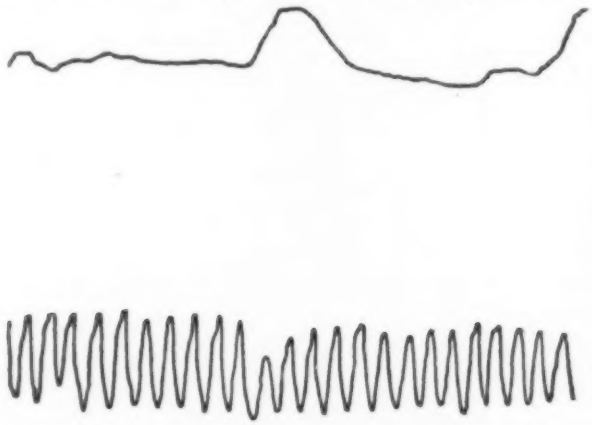

FIG. 4. — Expectation curve. Showing the changes in the electrical resistance and respiration due to expectant attention.

in Part II while the test-person is waiting for the fall of the weight. In height they vary as the reactions to the stimuli but are nearly always lower than these.

The average height of expectation curves is 15.70 mm. This high average is due to the fact that a test-person who has a great galvanic reaction to a stimulus will have many and great expectation curves. The time required for the curve to reach its maximum averages 10 sec., and to fall to the former level 12 sec.

The inspirations from the beginning to the top of the curve average 3.06 per cm., and during the fall average 3.3 per cm. The average respiratory amplitude during the rise is 10.18 mm., and during the fall 10.56 mm.

That the individual differences in the galvanic reactions are great will be seen by the average of distribution of the various averages.

Part II	weight	coefficient	8.09
Part IV	weight	"	8.71
Part IV	whistle	"	2.75
Part IV	picture	"	6.64
Part V	1. Sent.	"	4.7
	2. Sent.	"	4.42
	3. Sent.	"	7.63
	4. Sent.	"	3.98

This coefficient was obtained by taking the average of the sum of the differences between the average of all the figures and each figure. It shows, when large, that there is a great diversity in the numbers of which an average is taken, when low, that the numbers are nearly equal. Two of our test-persons had extremely high galvanic curves and therefore the average and coefficient is greater than would have been the case had these two cases been omitted. Our averages, on this account, are probably higher than other observers will obtain.

The pneumographic results are interesting because they differ from those obtained by other investigators and because they show a different relation between the rate and amplitude than one would expect.

The following table will show the averages of all the

averages of respiratory rate and amplitude and the average of distribution of each:

	Inspir. per cem.	Coeffi-cient	Height in mm.	Coeffi-cient
Before stimulus	2.94	0.16	12.19	0.62
Rise galv. curve	2.97	0.19	11.28	0.50
Fall galv. curve	3.11	0.13	12.19	0.47

As will be seen the respiratory rate increases from the moment of the stimulus while the amptitude decreases during the action of the affect and increases when it passes away. The coefficients in all cases are low, and show that the numbers of which an average was taken are about equal.

The relation between the respiratory rate and amplitude during the rise and fall of the galvanic curve and the high and low galvanic reactions is interesting. These relations were obtained by taking the averages of the sums of the respiratory rates and amplitudes of the high and low reactions of each individual before and after the stimulus.

They are:

During rise	high	rate	decrease 0.05 per cm.
		amplitude	decrease 1.17 per mm.
	low	rate	decrease 0.06 per cm.
		amplitude	decrease 1.06 per mm.

Thus during the rise the decrease in rate is practically the same in both high and low reactions but the decrease in amplitude is greater in the greater reactions.

During fall	high	rate	decrease 0.066 per cm.
		amplitude	increase 1.601 mm.
	low	rate	decrease 0.001 per cm.
		amplitude	increase 0.819 mm.

During the fall of the galvanic curve the rate decreases more in the greater than in the lesser reactions, while the amplitude also increases more in the greater than in the lesser reactions.

During the rise of the curve it is probable that part of the bodily innervation is expended on the various affective

muscular tensions, etc., and consequently the greater the individual reacts with other innervations the less will be expended on the respiration. This would explain the decrease in rate and amplitude in the greater reactions. During the fall of the galvanic curve the innervation is probably returned to the respiration but chiefly to the depth, the rate is slowed in some of the greater reactions.

The relations of the rate and amplitude before and after the reaction show that there is an increase in the rate and amplitude after high reactions and a decrease in the rate and increase in the amplitude after low reactions.

The following table was obtained by comparing the rate and amplitude before the stimulus with the rate and amplitude during the rise of the galvanic curve and the rate and amplitude during the fall of the galvanic curve with that during the rise of the galvanic curve.

Before stimulus	High	rate	increase 0.156 per cm.
		amplitude	increase 0.213 mm.
After reaction	Low	rate	decrease 0.091 per cm.
		amplitude	increase 0.093 mm.

This table shows clearly that the differences in the respiratory changes are much greater in the cases of the higher galvanic reactions.

As far as could be determined there was no regular relation between the height of the galvanic reactions and the individual bodily resistance at the beginning of the experiment.

ABNORMAL TEST-PERSONS

These test-persons consisted of patients suffering from epilepsy, dementia praecox, general paralysis, chronic alcoholism and alcoholic dementia and senile dementia.

The conditions of the experiment are exactly the same as in the case of normal test-persons except that in many cases the pneumograph could not be used.

Epilepsy. There were nine test-persons in this group, the majority being in quite a demented condition. Included is one case of traumatic epilepsy on a basis of congenital

imbecility and one case of epilepsy with hysteria. One test-person was examined immediately after an attack of petit-mal. In this case the reactions to ordinary stimuli were slight or nil, but when threatened with a needle there was a galvanometric deflection of 20 mm. This change was very slow and the curve remained high for several minutes. The threat of a prick of a needle is a very strong stimulus and causes reactions in practically every case where dementia is not marked. In this case the whistle produced a fluctuation of 4 mm. and the weight one of 2.8 mm. The other stimuli were without effect. The latent time for the whistle was 5 sec. and for the needle 15 sec. It required 21 sec. for the curve produced by the needle to reach its maximum.

In this group the differences between the reactions to physical and psychical stimuli are more marked than in normal test-persons.

The quiet period in all cases shows little change. Only one test-person shows what could be considered as an expectation curve.

Five test-persons reacted to the falling weight, Part II. The reactions vary from 3.2 mm. to 35.6 mm. The greatest reaction was in the case of epilepsy and hysteria. The three cases not reacting were in a very demented condition. The averages for the cases reacting are,

Height of galv. curve	7.5 mm.
Latent time	2.25 sec.
Time to top	6.00 sec.

The pneumographic results are:

	Rate per cm.	Average height
Before	2.6	12.28
Rise of galv. curve	2.6	9.73
Fall of galv. curve	2.71	10.81

The galvanometric reaction is only about one-third as high as the normal. The pneumographic results are practically those of the normal cases.

Spontaneous speaking (Part III) could only be tried in three cases. In these cases there was a latent time averaging

2 sec., as contrasted with the normal cases where the curve begins to rise with the moment of speaking.

The results for three cases are:

Height of galv. curve	14.66 mm.
Latent time	2.0 sec.
Time to top	5.5 sec.

These results are less than in the normal test-persons.

The pneumographic results are:

	Rate per cm.	Average height.
Before	3.5	10.92
Rise of galv. curve	3.3	11.52
Fall of galv. curve	2.9	13.62

In the normal cases the amplitude decreases from the moment of stimulus; here it increases.

Part IV. Three physical stimuli, weight, whistle and picture failed to cause any reaction in three demented cases.

The results for five cases are:

	Weight	Whistle	Picture
Galv. curve	26.6 mm.	23.6 mm.	15.4 mm.
Latent time	2.3 sec.	3.5 sec.	2.83 sec.
Time to top	6.6 sec.	6.75 sec.	5.3 sec.

In the normal cases the reaction to the picture was greatest. That to the weight, the one calling up the fewest associations, causing the least.

The pneumographic results in three cases are as follows:

	Rate per cm.			Averange height.		
	Weight	Whistle	Picture	Weight	Whistle	Picture
Before	2.8	3.	2.7	8.05	8.23	8.34
Rise	2.5	2.96	3.6	7.1	9.37	6.51
Fall	3.11	3.1	2.9	6.74	8.38	8.03

In the normal cases the height is always less during the rise of the galvanic curve, here it varies very much.

Part V, sentences caused comparatively slight reactions in all cases. In four demented cases there were no reactions.

The results for four cases are:

	1. Sent.	2. Sent.	3. Sent.	4. Sent
Galv. curve	13.4 mm.	7.8 mm.	4.5 mm.	4.5 mm.
Latent time	3.0 sec.	3.3 sec.	5.0 sec.	3.0 sec.
Time to top	3.6 sec.	5.0 sec.	5.0 sec.	2.0 sec.

The reactions decrease in intensity from the first to the third sentence.

The pneumographic curves give the following results:

Rate per cm.

	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Before	3.5	3.0	3.0	4.0
Rise	4.0	3.0	3.0	3.0
Fall	3.1	3.3	3.3	2.5

Average height in mms.

	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Before	7.2	6.7	5.6	7.0
Rise	6.1	7.5	6.0	5.5
Fall	6.8	6.0	6.6	5.5

Part VI. The second quiet period shows nothing.

In all these cases of varying degrees of dementia the galvanic fluctuations were in direct relation to the degree of mental dulling, the very demented having little or no reaction. In very demented cases the reactions are similar to those of the person cited above after an attack of petit mal, only those stimuli tending to cause pain are reacted to. The problem of this phenomenon is entirely a question of lack of associations.

DEMENTIA PRAECOX

The cases in this group were in various stages of the disease. The reactions, therefore, vary very much in the different cases. Each form of the disease will be discussed separately.

CATATONIA

There were eleven cases of catatonia varying from those in complete stupor to those in a convalescent condition.

Our results are high because one convalescent gave reactions which were those of a normal person. Cases in a condition of stupor give practically no reaction to ordinary stimuli and in those in a depressive state the reaction is also less marked.

The quiet curve varies according to the condition of the test-person. In patients who are actively hallucinated it is very often quite irregular; in patients in a stuporous condition it is practically a straight line.

The pneumograph was not used.

Part II (falling weight) caused a reaction in practically every case, the reaction varying from 1.8 mm. in a very depressed patient to 6 mm. in a patient with active hallucinations and 43.2 in a convalescent. The average deflection for eleven cases was 6.8 mm.

Part III (spontaneous speaking) was not possible with these test-persons.

Part IV (three physical stimuli) caused various reactions as in the normal cases. In five cases the whistle caused no reaction, the patients being in a stuporous, depressed condition.

The weight caused a deflection of 6.3 mm., the whistle 2.4 mm. and the picture 3.9 mm. As in the groups of epileptics the weight caused the greatest reactions.

Part IV (four sentences) in every case gave reactions less than the physical stimuli. The test-person who reacted to the weight with 43.2 mm. reacted to the sentences with a deflection of from 6 to 14 mm.

The averages for four sentences are:

1. Sent.	2.01 mm.
2. Sent.	2.3 mm.
3. Sent.	2.6 mm.
4. Sent.	1.9 mm.

The second quiet curve shows nothing.

HEBEPHRENIA

There were eleven test-persons suffering with this form of the disease. The results while not differing markedly

from those of the former group are quite different from the normal.

As in the former group, the quiet curve is irregular whenever the patient has marked hallucinations.

The weight (Part II) caused a less marked reaction than in the former group the average deflection being 5 mm.

Spontaneous speaking (Part III) in four cases gave an average deflection of 2.6 mm.

The three physical stimuli (Part IV) caused the following reactions: weight 6.8 mm., picture, 4.4 mm., and the whistle 3.5 mm. As in the former groups, the weight caused the greatest reaction.

Part V(sentences) causes a greater reaction here than in the former group but a much smaller average reaction than the physical stimuli. The results are:

1. Sent.	2.6 mm.
2. Sent.	1.3 mm.
3. Sent.	3.8 mm.
4. Sent.	4.2 mm.

PARANOID GROUP

There are four test-persons in this group, one in an early stage, two somewhat demented and one very demented. The latter reacted to none of the stimuli. The pneumograph was used in two cases.

The quiet period is practically that of a normal test-person.

Part II (falling weight) called forth reactions less than those in the two preceding groups, the average being 4.8 mm. The latent time averages 3 sec. and the time required for the curve to reach its maximum 7 sec. The rise and fall of these curves is much slower than in the normal cases.

The pneumographic results of two cases are:

	Rate per cm.	Aver. height in mms.
Before	2.5	13.1
Rise	2.94	8.1
Fall	2.63	11.8

These are practically the results obtained in the normal cases.

Part III (spontaneous speaking) was tried in two cases, giving an average deflection of 4.6 mm.

The pneumographic results are those of the normal cases.

	Rate per cm.	Aver. height in mms.
Before	3.2	11.78
Rise	2.92	9.2
Fall	2.52	10.76

Part IV (three physical stimuli) gives results which resemble those of the normal test-persons in that the reaction to the picture is the greatest.

The results are:

	Weight	Picture	Whistle
Galv. curve	5.8 mm.	7.0 mm.	5.4 mm.
Latent time	2.5 sec.	2.0 sec.	2.0 sec.
Time to top	6.0 sec.	5.5 sec.	6.0 sec.

The pneumographic results in regard to the depth of inspirations are practically those of the normal test-person. The rate varies in every case apparently without rule.

	Rate per cm.			Average height in mms.		
	Weight	Picture	Whistle	Weight	Picture	Whistle
Before	3.0	3.0	2.7	11.90	16.91	11.61
Rise	2.78	4.0	3.2	9.32	11.25	9.50
Fall	2.95	2.91	3.16	12.54	11.31	11.53

Part V. The reactions to the sentences are but little higher than those in the other forms of dementia praecox.

The results are:

	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Height	5.2 mm.	3.2 mm.	2.6 mm.	3.0 mm.
Lat. time	3.0 sec.	5.0 sec.	3.0 sec.	3.0 sec.
Time to top	4.5 sec.	5.0 sec.	2.0 sec.	1.0 sec.

The pneumographic curves for the first two sentences only are given, those of the other two being unfit for use.

	Rate per cm.		Average height in mms.	
	1. Sent.	2. Sent.	3. Sent.	4. Sent.
Before	3.2	3.2	12.52	13.58
Rise	3.16	2.99	12.16	12.1
Fall	2.5	2.48	13.	12.22

The second quiet curve is regular in all cases.

CHRONIC ALCOHOLISM

There are three cases in this group, confirmed alcoholics, but showing no dementia. The galvanometric results only are given. The reactions were fairly rapid and in the majority of instances were greater to all stimuli than were those of the normal test-persons.

The first quiet curve shows nothing.

Part II (falling weight) caused a deflection of 23.3 mm., greater than in any of the other groups.

Part III (spontaneous speaking) caused a deflection of 18.6 mm.

Part IV (three physical stimuli, weight, picture and whistle) caused the following deflections: weight 24 mm., whistle 24 mm., picture 28 mm. These reactions are greater than those of the normal test-persons. The relation of the reactions to the various stimuli in these cases and in normal cases is practically the same in all cases that to the picture being the greatest and those to the weight and whistle being practically the same.

Part V (the four sentences) caused reactions generally greater than in the normal cases, being, 1. Sent. 8.6 mm., 2. Sent. 16 mm., 3. Sent. 20 mm., 4th. Sent. 14 mm.

ALCOHOLIC DEMENTIA

There were three cases of alcoholic dementia which may be contrasted with the former group. In this group the reactions are all less than in the cases without dementia and especially striking is the lessened reactions to the psychical stimuli.

The weight caused a deflection of 9.06 mm. as compared to the 23.3 mm. of the former group.

Spontaneous speaking caused a reaction of 6.8 mm.

The reactions to the three physical stimuli, weight, picture and whistle are very interesting. The picture caused a deflection of only 7.6 mm., as compared to the weight 16 mm., and the whistle 13 mm. The reactions are directly proportional to the physical nature of the stimuli. The picture which in normal cases caused the greatest number of asso-

ciations and the greatest affects here causes the fewest associations and slightest reactions.

The reduction of the reactions to mental stimuli is again well seen in the sentences where they are slight.

1. Sent.	2. Sent	3. Sent.	4. Sent.
3.3 mm.	1.3 mm.	5.6 mm.	2.5 mm.

The lessening here is proportionally much greater than in any of the other groups.

GENERAL PARALYSIS

Nine cases of general paralysis were examined. Two were in a condition of euphoria and one in a period of remission. The other six cases were in a condition of dementia and apathy and gave practically no reactions to the various stimuli.

The quiet period in the cases of dementia shows nothing at all; in the other cases a few irregularities are seen.

Part II (the falling weight) caused good reactions in the two euphoric cases and the case in a remission but no reaction at all in the demented cases.

Galv. curve	21.1 mm.
Lat. time	2.2 sec.
Time to top	6.6 sec.

The pneumographic results in these cases are practically normal.

	Rate per cm.	Average height
Before	3.25	8.7 mm.
Rise	3.1	7.2 mm.
Fall	3.4	9.6 mm.

In the cases not giving a galvanic reaction the pneumographic results for two cases are:

	Rate per cm.	Average height
Before stim.	2.5	21.37 mm.
After stim.	3.0	22.3 mm.

Spontaneous speaking could not be attempted.

Part IV (three physical stimuli) in the three cases caused the following reactions:

	Weight	Picture	Whistle
Galv. curve	9.4 mm.	15.05 mm.	25.8 mm.
Lat. time	2.5 sec.	2.6 sec.	2.3 sec.
Time to top	4.0 sec.	4.1 sec.	7.0 sec.

The high average reaction to the whistle is due to the reaction of the patient in a period of remission whose reaction was 70 mm. It will be observed that the weight in these cases causes the slightest reaction.

The pneumographic results for the three cases are:

	Rate per cm.			Average height in mms.		
	Weight	Picture	Whistle	Weight	Pioture	Whistle
Before	3.0	3.65	3.0	5.5	7.9	5.5
Rise	3.0	3.2	3.0	4.5	7.8	9.1
Fall	3.0	3.5	2.9	4.8	7.8	8.8

In the case of two patients not giving a galvanic reaction the pneumographic results are:

	Rate per cm.			Average height in mms.		
	Weight	Picture	Whistle	Weight	Picture	Whistle
Before	2.0	2.0	3.0	20.5	18.5	20.45
After	2.0	2.0	2.5	21.12	19.0	20.50

Part V. The results for three sentences are given. Four test-persons reacted to these stimuli.

	1. Sent.	2. Sent.	3. Sent.
Galv. curve	16 mm.	9.58 mm.	18 mm.
Latent time	4 sec.	2.5 sec.	1.5 sec.
Time to top	5 sec.	4.7 sec.	5.5 sec.

These reactions are practically the same as those of the normal test-persons.

The pneumographic results for these cases are:

	Rate per cm.			Average height		
Before	3.5	3.0	3.0	7.4 mm.	7.1 mm.	10.3 mm.
Rise	4.0	3.3	3.3	10.0 mm.	8.6 mm.	9.0 mm.
Fall	4.0	4.6	4.5	11.0 mm.	8.1 mm.	9.2 mm.

The pneumographic results of two cases not giving a galvanic reaction are:

	Rate per cm.		Average height in mms.	
	1. Sent.	2. Sent.	1. Sent.	2. Sent.
Before	2.75	3.0	20.75	20.40
After	2.75	2.75	21.30	21.50

Paretics in a condition of euphoria and in a stage of remission, when dementia is not pronounced, react well to the various stimuli. They take a very active interest in the experiment, and this may account for the fairly large galvanic reactions. Paretics in a demented condition give no reactions to simple stimuli and correspond to other dements.

SENILE DEMENTIA

There were eleven cases of senile dementia. In these cases the great majority did not react to the stimuli. In some cases even the prick of a needle caused no galvanic fluctuation.

The weight caused a reaction in three cases. The average deviation for the three cases was 5 mm.

Spontaneous speaking could not be attempted on account of the dementia.

The three stimuli (Part IV) gave results which are less than those obtained in any other disease, the weight causing an average deflection of 1 mm., the whistle 1.8 mm., and the picture 4 mm. The relatively high reaction caused by the picture is interesting.

The mental stimuli, sentences (Part V) cause very little reaction.

1. Sent.	2. Sent	3. Sent.	4. Sent.
0.6 mm.	0.6 mm.	0.2 mm.	0.8 mm.

The following table gives a collective view of the galvanic results of all the test persons.

	Wght.	Spontaneous	Wght.	Whistle	I. Sent.	2. Sent.	3. Sent.	4 Sent.	
Normal	20.6	17.9	17.94	19.72	18.2	14.62	14.48	19.92	11.12
Epilepsy	7.5	14.66	26.6	15.4	23.6	13.4	7.8	4.5	4.5
Catatonia	6.8	—	6.3	3.9	2.4	2.01	2.3	2.6	1.9
Hebephrenia	5.0	2.6	6.8	4.4	3.5	2.6	1.3	3.8	4.2
Paranoid D. P.	4.8	4.6	5.8	7.0	5.4	5.2	3.2	2.6	3.0
Chronic Alcohol	23 .	18.6	24.0	28.0	24.0	8.6	16.0	20.0	14.0
Alcoholic Dementia	9.06	6.8	16.0	7.6	13.0	3.3	1.3	5.6	2.5
General Paralysis Euphoria and remission	21.1	—	9.4	15.5	25.8	16.0	9.5	18.0	—
General Paralysis Dementia	—	—	—	—	—	—	—	—	—
Senile Dementia	5.0	—	1.0	4.0	1.8	0.6	0.6	0.2	0.8

The above table shows that in every case the physical stimuli cause a less galvanic fluctuation than do the psychical but in the cases where intellectual deterioration is marked the reduction is proportionally greater than in the other cases.

The intensity of the reaction seems to depend in part on the attention paid by the test-person to the experiment. In cases of dementia praecox where the internal complexes dominate the affectivity and attention the reactions are slight; in alcoholism and in general paralysis, euphoric state, where the excitability is very great, the reactions are correspondingly greater. In organic dementia where all associative power is lost the reactions are almost nil. In dementia senilis where dementia was very marked even a prick of a needle failed to cause a response.

The pneumographic results in these cases are practically those found in normal cases. There is evidently no rule for the rate but as a general thing the amplitude decreases during the action of the galvanic phenomenon.

That the galvanic fluctuation is caused by the psychical and not the physical factor of a stimulus is shown by the following facts:

The reaction is greatest when the stimulus is such as to call up a great number of associations, *e.g.*, the picture.

A stimulus which causes doubt and perplexity is accompanied with a marked galvanic fluctuation, *e.g.*, where the stimulus is a simple word.

In cases of dementia where associations are few the reactions are correspondingly decreased.

The physical intensity of a stimulus does not bear any regular relation to the size of the galvanic reaction.

The strength of the reaction changes exclusively along psychological constellations. This is shown beautifully in one normal case where an ordinary whistle caused but a small reaction, but the whistle call of the society to which the test-person belonged when he was in school caused a very great galvanic fluctuation.

If the attention is not directed to the stimulus the reaction is small or nil. Therefore we have no reactions in those cases where the attention is seriously disturbed. This can be proven by letting the test-person count or make lines on a paper at the stroke of a metronome. In this case the reactions are practically nil.[1]

SUMMARY

From the above experiments we conclude that:

1. The galvanic reaction depends on the attention to the stimulus and the ability to associate it with other previous occurrences. This association may be conscious but is usually subconscious.

2. Physical stimuli as a rule cause greater galvanic fluctuations than do the psychical in our experiments. This

[1] Disassociation Experiments, by Binswanger. Jungs' Diagnotische Assoziationstudien, XI. Beitrag.

may be due to the fact that they occurred before the psychical stimuli, early stimuli nearly always causing greater reactions than do later ones.

3. While the normal reactions vary greatly in different individuals, they are as a rule always greater than pathological reactions.

4. In depression and stupor the galvanic reactions are low because the attention is poor and associations are inhibited.

5. In alcoholism and in the euphoric stage of general paralysis the reactions are high because of the greater excitability.

6. In dementia the reactions are practically nil because of the lack of associations.

7. The reactions show great individual variations and within certain, rather wide, limits are entirely independent of the original bodily resistance.

The pneumographic results may be summarized as follows:

1. The inspiratory rate varies according to the individual and no general rule can be given.

2. The amplitude of the inspirations is generally decreased during the rise of the galvanic curve.

3. This decrease in the amplitude, however, has no relation to the height of the galvanic curve but varies according to individuals.

4. In cases of dementia where there is no galvanic reaction the changes in the respirations exist but are very slight.

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MECHANISM OF A SEVERE BRIQUET ATTACK AS CONTRASTED WITH THAT OF PSYCHASTHENIC FITS

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FRESH interest has been aroused of late, chiefly through the writings of certain German and French workers, in the important group of conditions characterized by transitory affections of consciousness. The older view that only a broad use of the term epilepsy was needed in order satisfactorily to include all such conditions had the merit of simplicity, but not of accuracy. Although the terms psychic epilepsy and petit mal have done hard service in the past, it is now widely recognized that there is a large group of conditions often incorrectly diagnosed under these titles, and particularly under the former. The major section of this group belongs to the psychoneuroses and is not of an epileptic nature. It is not my purpose here to discuss the separation of the epilepsy group from the psychoneuroses, but merely to consider a point in the differentiation of some of the cases that admittedly belong to the latter.

A few words on terminology are first necessary. Oppenheim[1], in his recent important contribution, uses the expression psychasthenic convulsions (Krämpfe) in describing certain attacks characterized by loss of consciousness with or without convulsive movements. Spiller[2] points out that this is too limited an expression, and prefers the term psychasthenic attacks. It might be objected that, unless it is further qualified, this term on the other hand is too wide, as it is applied in psychopathology to widely different mani-

1 Oppenheim, Journ. fur Psych. u. Neur., 1905-6, Bd. VI, S. 247.

2 Spiller, Journal of Abnormal Psychology, February, 1907, Vol. I, p. 257.

festations, both psychical (as Janet's psycholeptic crises[1]) and physical (as recurring pareses.) The word attack suggests to the mind two features of the condition under discussion, its sudden or rapid onset, and its temporary duration. As however there is a third constant feature, namely, affection of consciousness — of all degrees, from slight obnubilation to deep coma — it would be desirable to indicate this also, if possible. The word "fit" embodies the three characteristics common to all the conditions now referred to, and has the further advantage of being the term already in use in connection with two of the most important varieties, namely apoplexy and epilepsy. Janet[2] has applied the designation "Briquet attacks" to the more common hysterical crises, in contradistinction from the elaborate performances described by Charcot. Hystero-epileptic fits would be included under this designation, for, although their existence had long before been clearly appreciated by Louyer-Villernay,[3] Landouzy[4] and others, it was Briquet[5] who first gave us a systematic account of their clinical varieties.

In the diagnosis of the nature of the fits we may collect evidence from observation of the fit itself, or from examination of the patient during the interval period. Considering first the fit itself, we may be able to make an immediate diagnosis of hysteria if the symptoms displayed are those of the classic hysterical fit, the only one described by most text-books as occurring in hysteria. If however the fit is of an epileptiform variety — either grand mal or petit mal — the matter is very different. Heilbronner[6] has remarked that in this variety the difficulty of diagnosis, so far from diminishing with our improved knowledge of the underlying conditions, has considerably increased, chiefly owing to the recognition that more phenomena are possible in hysteria than was previously conceived. It is

[1] Janet, Boston Med. and Surg. Journal, Jan. 26, 1905, P. 93.
[2] Janet, Mental State of Hystericals, New-York, 1901, P. 369.
[3] Louyer-Villernay, Traite des Maladies nerveuses, Paris, 1816.
[4] Landouzy, Traite complet de l'Hysterie, Paris, 1846, P. 144.
[5] Briquet, Traite clinique de l'Hysterie, Paris, 1859, P. 327 et seq.
[6] Heilbronner, Ueber gehaufte kleine Anfalle. Deutsche Zeitschr. fur Nervenheilk., 1906, Bd. XXXI, S. 472.

becoming recognized that in a grand mal attack there may be absolutely nothing in the nature of the attack itself to indicate its source. Hoche[1] has shewn that one after another the various features that had been advanced as distinguishing an epileptic from a hystero-epileptic fit have been proved to be non-pathognomonic, until at present it is definitely known that every symptom of a grand mal fit, from fixed pupils to sphincter relaxation, may occur as well in functional affections as in idiopathic epilepsy. The features referred to are of course more frequent in the latter condition, so that the value of their observation is diminished rather than nullified.

It follows from the foregoing that while in many varieties of fits it is possible to exclude epilepsy, it is hardly ever possible to exclude hysteria from observation of the fit alone. As a result of this advance in knowledge the modern tendency has been to base the differential diagnosis in doubtful cases upon an investigation of the mental state of the patient during the free interval rather than upon the symptoms displayed in the fit itself. Heilbronner,[2] for instance, refuses to diagnose epilepsy in the absence of the characteristic mental changes found in that condition apart from the fits. In the differentiation and analysis of hysteria during the interval period, Jung[3] and Ricklin[4] have obtained very striking results by the application of the Association-reaction method. Without the use of some exact method of investigation such as Jung's[5] there is of course the great difficulty that two conditions such as hysteria and epilepsy may coexist, and some authorities, including Binswanger,[6] maintain that these two may pass over from one into the other with no sharp dividing line, a view of great theoretic and prac-

1 Hoche, Die Differentionaldiagnose zwischen Epilepsie und Hysterie, Berlin, 1902, S 18.

2 Heilbronner, loc. cit.

3 Jung, Journ. fur Psych. u. Neur. 1902-3, Bd. I. S. 110.

4 Ricklin, Die diagnotische Bedeutung der Assoizationsversuche bei Hysterischen. Psychiatr.-neurolog. Wochenschr., 1904. Nr. 29, Zur Psychologie hysterischer Dammerzustande. Ibid, Nr. 21, v. 22, 1904. Analytische Untersuchung der Assoizationen eines Falles von Hysterie, Ibid. Nr. 46-52, 1905. Kasuistische Beitrage zur Kenntniss hysterischer Assoziationsphanomene. Journ. fur Psych. u. Neur., 1906, Bd. VII, S. 223.

5 Jung. Journ. fur Psych. u. Neur., Bd. III. u. IV.

6 Binswinger, Art. Die Hysterie, in Nothnagel's Spec. Path. u. Ther., Bd. XII, Ht. 1: Thl. 2, S. 835.

tical interest. In children another difficulty is introduced by the frequency with which the psychoneuroses appear in a monosymptomatic manner, and often as purely psychical manifestations, similating for instance petit mal. It is especially under these circumstances that a point of view to which I wish to call attention may become of value, namely the study, through the subconscious memory, of the immediate cause of a given fit. Putnam and Waterman[1] remind us that our knowledge of the subconscious memories of epileptics is at present *nil*, and that by the study of them closer resemblances may be found between the subconscious states of hysterics and epileptics than we imagine. Much work on this subject has aleady been done by the Zurich school, particularly by Gräter,[2] Muralt,[3] and Jung,[4] while on the other hand supposed cases of psychic epilepsy have by this method been proved by White,[5] Parker,[6] and others to be really cases of major hysteria. Naturally when, as in the latter cases, a disaggregated mental focus has by this means been shewn to be the exciting cause of the fit, the diagnosis of epilepsy becomes thereby excluded. That the same method may prove equally fruitful in the differentiation between hysteria and psychasthenia, a differentiation that is so important on account of their widely different prognosis and treament, I hope to be able to shew by the study of the following case which was recently under my care.

Wm. C., a man-of-war's man aged eighteen, was brought to the Seamen's Hospital, Greenwich, at eight in the morning on March 4. He was then, according to the house surgeon's account, able to walk when supported on either side but seemed very dazed. He was trembling violently, could not reply to questions, and appeared not to appreciate his surroundings. He had been found unconscious in a garden, had gradually aroused and had been immediately assisted to the hospital. There were no abnormal signs in the nervous system.

[1] Putnam and Watermann, Boston Med. and Surg. Journal, 1905, Vol. CLIII, P. 509.
[2] Grater, Zeitschr. fur Hypnotismus, 1899, Bd. VIII, S. 129.
[3] Muralt, Zeitschr. fur Hypnotismus, 1905, Bd. X, S. 75.
[4] Jung, Journ. fur Psych. u. Neur., 1905, Bd. V. S. 73.
[5] White, Psychopathological Researches. Sidis, New York, 1902, P. 123.
[6] Parker, Ibid.

His state gradually improved in the course of the next hour and he then went to his home near by, returning at 10.30, when I saw him at my visit to the hospital.

At that time he was still trembling and frightened, and complained greatly of severe headache. This he described as a "very unpleasant, dizzy feeling, shooting backward from the top of the head." His hands were very tremulous, but I could elicit no other abnormal physical signs. He told me that he remembered dressing that morning about five o'clock with the intention of catching an early train to Chatham where his ship lay, but that he remembered nothing further until nine o'clock, when he felt revived by going out of the hospital into the open air. From his brother I learned that he was a healthy boy, not particularly nervous, and had never suffered from any similar attack. There was no psychopathic family history.

As his pain was so severe, I asked him to lie down and closed his eyes. His limbs almost at once relaxed, and he began to talk — reluctantly at first — in a quiet monotone. He then disclosed in a rambling fashion the following details, which I have pieced together more connectedly:

There had been some alienation between the boy and his mother, of whom he was very fond, on account of his entering the Navy in spite of her disapproval. It abated, however, as time went on, and he used to pay monthly visits of a couple of days' duration to his home. The last one of these was on February 1. In the middle of February his mother who had previously been in perfect health was seized with pneumonia and died in a couple of days. He could not get leave in order to visit her, neither was he able to attend her funeral. He was particularly distressed by this latter fact and made the curious observation: "If I hadn't gone to sea I should have been able to attend her funeral." On Saturday, March 3, he went home for the week-end, and spent a great part of the time talking about the recent event. Sunday night he sat up with his father and brother till after midnight talking thus. In the night he had a number of dreams about his mother. These seem to have been momentary pictures of her under various cir-

cumstances, but in several dreams there was movement, in which he and his mother played a prominent part. Some were pleasant, some indifferent, and one was exceedingly distressing. In this last he appeared to be returning home from a voyage, and on entering the living-room was repulsed by his mother, whose coldness produced in him great anguish. He woke at this point and heard a clock strike four. He again slept but got up just before five. During dressing he was reflecting on the dreams. As soon as he had dressed he left the house, with the intention of catching the early train. At this time dawn was just breaking and objects were dimly visible through the mist. As he left the front door, on his way down the garden path, he caught sight of an apparition of his mother standing some yards away at the gate. It was extraordinarily vivid and clear and he observed every feature of her dress, including a shawl he had given her, her rings, earrings, etc. She looked fixedly at him, a little reproachfully, but did not move. He was intensely frightened and turned to run into the house. In the act of turning everything "went red," and he fell unconscious on to his face. He could not recover further memories of any occurrence up to the time when some water was dashed in his face by his brother, who found him lying motionless on the path. This was at 7.45, more than two hours later. His memory for further happenings, including the short journey to the hospital and the interviews there with the nurses and house surgeon, was moderately complete; during this time he had been conscious of little besides terror, which "made him shiver."

Having reached this point in the investigation of the case, I recognized that I was in the presence of a very unusual opportunity. Here was a case of major hysteria in the making. A definite focus of disaggregation, invested with a powerful emotion-complex, had been rapidly growing in the past few days and had reached the surface for the first time only a few hours ago. If the boy were merely allowed to recover from his crisis and to return to work, we might expect within perhaps a few weeks some major manifestation of hysteria to develop, possibly paraplegia, blindness or

hystero-epilepsy. Recovery from the crisis meant merely the submergence of this dangerous focus, which sooner or later would again exert its devastating influence on the mind. Fortunately however seeing the case so clearly in the process of disaggregation gave an opportunity to reunite the focus to the main mental content and so prevent further insulation of the powerful body of emotion. I therefore employed a little suggestion along obvious lines, including future memory of all the events above detailed, and woke the patient up. He now felt completely well, was quite composed and clear, with a perfect memory of the recent events, and had no headache. He told me that he with several comrades had once been "mesmerized" in India by a native woman. It is hard to say whether the state that he was in when he recounted the above details was a hypnoidal or hypnoidic one — to use Sidis' terms,[1]— though I incline to the latter view. It certainly was not a hypnotic one in the ordinary sense of the word.

Let us now consider the value that the study of cases like the above has for diagnosis in general. In this particular instance it was fairly evident from the first that we had to do with a functional condition, though of course post-epileptic coma and automatism had to be carefully considered. From what was revealed later it was clear that the unconsciousness during the first two hours was quite profound, although which variety of hysteric coma was present is a matter of only conjecture. The next problem was to decide, if possible, which of the two chief psycho-neuroses — hysteria and psychasthenia — was present. The subsequent recovery of the submerged memories was of the utmost value in the determination of this question, for there is no doubt that the discovery of a single disaggregated focus, having a strong emotion-complex as its centre, was important evidence in favor of the hysterical nature of the condition. The reason why this was so is as follows:

Although from one point of view psychasthenia is excellently summarized in Janet's conception of it as a "lowering of the psychological tension[2]", its pathogeny can

[1] Sidis, Multiple Personality, New York, 1904.

[2] Janet, Les Obsessions et la Psychasthenie, Paris, 1903. La Revue Idees, Oct. 15, 1905, p. 729.

perhaps best be described in terms of chronic disaggregation, as Donley[1] for instance has done in the case of neurasthenia. Nevertheless there are certain precise differences between this disaggregation and that of pure hysteria, the two most important being perhaps the following. In the first place hysterical disaggregation has the striking characteristic of what may be termed cohesiveness, that is to say that the primary disaggregated focus shews a marked tendency to attract to itself similar foci, whether from the main content of the mind or from fresh experiences that have not proved capable of being assimilated. This tendency of the various foci to cohere, and to grow at the expense of the main body of mental content, is of course the explanation of the development of secondary personalities, and the stages by which the incomplete forms of this shew the first signs of a separate self-consciousness provide studies having important bearings on the theory of consciousness in general. A further characteristic of the hysterical disaggregation is its dependence on a single original focus, caused by either physical or psychical trauma; effective dealing with this focus means the disappearance of the disease. The disaggregation of psychasthenia contrasts with that of hysteria on both these points. It never grows steadily as a unitary product outside the main mental content, and so never leads to the formation of secondary personalities, nor so frequently as hysteria to striking physical manifestations. Further its origin is rarely in one single focus that can be dealt with, but in a large number, a fact that renders radical treatment much more difficult. One might summarize some of the aspects of the problem in the statement that the disaggregation of hysteria is massive, while that of psychasthenia is molecular.

The matter is however much more complex in fact because of the extreme frequency with which primary hysteria is complicated by psychasthenic symptoms; the reverse is very rare. So usual is this occurrence that pure hysteria is an exceedingly rare disease; it may be doubted whether there are half a dozen cases of it on record. Morton Prince[2] has recently drawn attention to the ease with which

[1] Donley, Journal of Abnormal Psychology, June, 1906, P. 497.

[2] Boston Med. and Surg. Journal, Oct. 4 and 11, 1906. Vol. 4 CLV. P. 372 and 407. Journal of Abnormal Psychology. Oct. 1906. Vol. 1. P. 170.

hysterical disaggregation, so massive as to have gone on to the formation of multiple personalities, may be overlooked on account of the case presenting itself with the clinical picture of neurasthenia. We thus see that hysteria is more likely to be overlooked than is psychasthenia, so that definite evidences indicating the presence of the former condition are more significant than are evidences of the latter, which if present may or may not be of a primary nature.

To revert to the subject of the diagnosis of a fit of psychic origin, it becomes all-important to try to discover the nature of the disaggregated focus, and one method of achieving this is based on a study of the immediate mechanism of an individual fit. In the case above described, the unitary and relatively simple nature of the disaggregated focus plainly indicated its hysterical nature. It is, by the way, interesting to note its relation to dream states in this instance, in view of the importance attached by Freud[1] to dreams in this connection. The second great characteristic of hysterical disaggregation, namely its tendency to extend, will I trust never be manifested in this instance, for the uniting of the recently formed focus to full consciousness before it had time to establish itself securely has ended its existence as a disaggregated complex. The boy has remained perfectly well up to the time of writing.

I have mentioned here no psychoneuroses other than hysteria and psychasthenia, because it is not at present proved that any other group exists apart from these. Raymond[2], in his recent comprehensive survey of the subject, refuses to admit any other member. Strenuous attempts have been made, for instance, to create a separate group entitled Narcolepsy, by many writers from Gélineau[3], who first used this term, to Friedmann[4], who has recently given us a critical review of most of the recorded cases. It is granted that this syndrome may, in many cases at least, be symptomatic of hysteria or psychasthenia. In fact, several of the earliest

1 Freud, Traumbedeutung. Wien. 1900.

2 Raymond, Nevroses et psycho-nevroses, L'Encephale. Partie neurologique, Janvier, 1907, P. 1.

3 Gelineau, De la Kenophobie, Doin, Paris, 1880. De la Narcolepsis, Paris, 1881.

4 Friedmann, Ueber die nicht-epileptischen Absencen oder kurzen narkoleptischen Anfalle. Deutsche Zeitschr. fur Nervenheilk. Juni 1906, Bd. XXX. S. 462.

cases were described in connection with kenophobia, or, to use the more customary expression invented by the Germans, agoraphobia. In other cases, chiefly in children, there are no other sympoms present. This however, as Heilbronner[1] points out, in no way proves that the syndrome cannot be grouped under the above two psychoneuroses, which unquestionably are often monosymptomatic, particularly in children. There can be no justification for the establishment of a psychoneurosis group apart from hysteria and psychasthenia, until the psychological mechanism of such a group has been shewn to be different from that of these two conditions.

To sum up, in considering the differential diagnosis of fits of doubtful nature, besides the careful observation of the fit itself, and the investigation of the mental state in the interval periods, we may sometimes be able to obtain valuable indications from a study of the mechanism initiating an individual fit.

CRITICAL REVIEWS

MOTOR APHASIA AND ITS CORTICAL LOCALIZATION. *By J. Dejerine, L'Encephale, May, 1907.*

In this article of fifty pages Dejerine returns to the contest with Marie over the doctrine of aphasia. The author undertakes the task, not only of upholding his previous view,— the classical view — in favor of the third frontal convolution being a motor speech centre, but of determining the limits of this centre, *i.e.*, whether the centre of language is limited to the foot of Broca's convolution, or whether the neighboring regions take part in this function. It is only by serial section, it is maintained by him, that this can be done. Even the most minute "cortical lesions" are more extensive than they appear to be from inspection of the cortex. This is true of them both superficially and in depth. The neighboring convolution, which appears to be normal, will generally be found to have its white matter scooped out by extension of the lesion. True cortical lesions are always subcortical as well. Much more frequent still are lesions which fuse in the centrum ovale at a considerable distance, cutting

[1] Heilbronner, Loc. cit., S. 476.

the fibres which issue from more or less distant convolutions; or, penetrating into the depth of the hemisphere, destroy the central gray nuclei and internal capsule. The lesions often send diverticula irregularly in different directions. The interpretation of the symptoms therefore depends on these conditions which can be determined only by serial sections.

Dejerine repeats his former statement that while it is certain that in the third frontal convolution is a motor centre for language, it has not yet been proved that this centre is sharply limited to the *foot* of this convolution. It is possible, but not yet proved. Future researches may extend the site so as to take in the neighboring cortex, excepting that the adjoining operculum, whose functions are known, can be excluded. (Bernheim had suggested that the limits might be extended to the foot of the second frontal and to the anterior convolutions of the insula.) The same criticism may be made of the exact limitation of sensorial centres.

To date there has been no case of aphasia in which a lesion limited to the third frontal convolution and the underlying white matter, *without extension of the lesion to the white substance of neighboring regions*, has been studied by the method of serial sections.

The author now reports two such cases. The *first* was one of lesion of the anterior two-thirds of Broca's convolution. The symptoms were complete loss of the function of speech, without paralysis of the muscles, and without loss of comprehension of spoken or written language. As to the latter, however, long phrases were not understood, and it does not appear that any but simple tests, such as commands to execute such and such a movement, or to take such and such an object, were used to test the power to comprehend spoken language. Mimicry was preserved. At the end of five months speech returned, though until his death, two years later, the patient "exhibited a certain slowness in the evocation of many words, and he spoke in short phrases, while stopping and beginning again."

The fact that this patient nearly completely recovered the faculty of speech weakens the case as evidence of localization, for it is plainly open to the interpretation that the neighboring regions, the insula for instance, or even Marie's somewhat

mystical "lenticular zone," might take part in or be the real centre for motor speech, and be temporarily involved by vascular (oedema) or other disarrangement. One cannot help regretting that the clinical study of this case was so brief, the report occupying about two-thirds of a page, while such an enormous amount of work was given to the anatomical study. This criticism applies also to the second case. We need careful clinical studies of aphasia quite as much as anatomical studies if we are to solve the riddle of language.

The second case was one of lesion of the head of Broca's convolution. There was loss of the power of speech, but retention of comprehension of spoken language including complicated orders. Reading was affected, so that the patient could understand only isolated words and short familiar phrases. She could not write spontaneously or to dictation, but could copy correctly. Mimicry was preserved, and memory was good.

There was no recovery of the lost functions. This case, therefore, is more valuable for localization than the first. Numerous illustrations of sections of the brain showing the extent of the lesions in both cases are given in the text. "I have no need of insisting," the author remarks, "upon the documentary importance of these two autopsies. . . . [They] demonstrate that *motor* aphasia, called *cortical*, or *Broca's*, can be produced by a lesion limited to the *anterior part* of the zone of language, that is to say, to the *zone of Broca* — without any participation, so far as concerns the primitive lesion (whether on the surface or underneath) of the Rolandic operculum, of the motor convolution, of the zone of Wernicke (temporal cortex and underlying white substance), or of the central gray nuclei.

"They demonstrate that there exists in the *left frontal lobe a zone, the zone of Broca, a lesion of which produces aphasia, and this without any alteration of the temporal lobe, of the external and internal capsules, of the central gray nuclei and the motor convolutions.*" [p. 492]

While such evidence is positive regarding the function of a particular region of the cortex, Dejerine, in making the point of the non-participation of other neighboring parts in the lesion, and therefore in the zone of language, seems to forget that such evidence in this respect is negative and is not proof. The

destruction of a portion of a centre may well produce sufficient disturbance to abolish function.

Dejerine next enters into a renewed discussion of what will be regarded as the most important issue in this aphasia question, namely, the doctrine as maintained by Marie. His argument is presented with force and his criticisms of Marie are decidedly effective. He takes up the evidence upon which Marie relies in support of the contention that the third frontal convolution has nothing to do with language which, as a faculty, is located alone in Wernicke's zone. Marie reports three cases with autopsies, all right-handed people, who with lesion of the third frontal convolution were not aphasic at the time of death; but, according to his critic, information is lacking regarding the condition of articulate speech in the earlier years. It is well known that not rarely motor aphasia clears up (Dejerine's first case is an example); consequently at the autopsy we may find a lesion occupying Broca's convolution alone, as in the first of Marie's cases, or both this convolution and Wernicke's zone, as in his last two cases. In the last two cases it is equally remarkable that these patients did not exhibit sensorial aphasia. Dejerine refers to cases in the literature of patients with motor aphasia which disappeared, although at the autopsy lesion of Broca's convolution was found. Such cases may be explained on the ground that the subject was ambi-dextrous, or on that of the coöperative functioning of the homologous convolution on the right side. A case in evidence of this is cited. Dejerine makes the point that all the cases of Broca's aphasia reported by Marie were cases either of *total aphasia* or *sensorial aphasia*. Consequently, it was quite natural that the sensorial (Wernicke's) zone was injured. Here it should be pointed out that much confusion has arisen from Marie and Dejerine obstinately persisting in using the same term, Broca's, for entirely different syndromes, and then wrangling over the localization of the term. It is the syndrome and its anatomical correlate, not the name of it, with which we are concerned for the purpose of forming a doctrine of aphasia. By Broca's aphasia, Dejerine means the classical motor type, with loss of internal language, without paralysis, and without impairment of the comprehension of language; but Marie means thereby the impairment of the comprehension of language plus

a motor type which he chooses to name anarthria. Though the difference in these clinical syndromes and in the use of the term "Broca's aphasia" is insisted upon by both writers, nevertheless, by using the same term for different syndromes, and the substituting by Marie of anarthria for aphasia, considerable dust is raised, which obscures the discussion.

The first problem then is, whether there is a motor type, what is its syndrome, and what is its location?

Both Dejerine and Marie admit a motor type, but Dejerine postulates two types, viz.:

A. Loss of articulate language, without paralysis and without loss of comprehension of spoken and written language, but *with* loss of internal language. It is localized in the third frontal convolution.

B. The same syndrome, but *without* loss of internal language.

This second type, B, is identical with Marie's type (as Marie admits), only Dejerine calls it *pure* motor aphasia, and gives it a subcortical localization in the centrum ovale, while Marie calls it *anarthria*, and locates it rather vaguely in the lenticular zone.

Here then we have on the motor side the difference and agreement in the two views. Marie denies A, and that the third frontal convolution has anything to do with language. He admits B, with a moderate difference in the localization.

Now taking aphasia so far as it is a *loss of the comprehension of language*, both admit this type and agree to its localization in substantially the same place in the cortex, viz., Wernicke's zone — only Marie attributes the loss of comprehension of language to a "special intellectual defect, a failure in elaboration"; while Dejerine holds to the classical notion of loss of sensory images.

[This it will be noticed is a pure psychological question, though Marie pretends to spurn psychology and imagines he is dealing with simple clinical conditions! whatever that may mean.]

Now both Dejerine and Marie agree that these two clinical defects, the motor defect and the loss of comprehension of language may be combined, in which case we have what Dejerine

calls *total* aphasia, and Marie, *Broca's* aphasia, and over this difference in names blood has nearly been spilt, while the syndrome for which the names stand has been left to shift for itself. Let us compound the names; then for Marie, "total — Broca's" aphasia equals loss of comprehension of language plus *pure* motor aphasia (B); for Dejerine, "total — Broca's" aphasia equals loss of comprehension of language plus motor aphasia (A). The difference lies in the alleged fact that there is loss of internal language in motor, but not in pure motor aphasia.

Corresponding to the two clinical defects there are two anatomical defects; for Marie these are located in Wernicke's zone plus the (motor) lenticular zone; for Dejerine, in Wernicke's zone plus the (motor) third frontal convolution.

It would follow that the loss of *internal* language, according to Marie's doctrine, would be due to the injury of Wernicke's zone; and according to Dejerine's, to that of the motor zone.

When thus stated the fundamental issues underlying this controversy become clearer. The main issue, then, is whether the third frontal convolution is a motor centre for language, or whether this centre lies in the "lenticular zone." A subordinate issue is whether in either case loss of internal language follows a destruction of this centre whichever it may be. Let us take the main issue first. Marie has modified his previous view that the motor lesion may be in either hemisphere and places it in the left hemisphere alone in a "quadrilateral" situated behind the third frontal convolution, limited antero-posteriorly by two lines, one drawn from the anterior, and one from the posterior limit of the insula. The external boundary is the insula, and the internal, the ventricular wall. Dejerine criticizes this localization in that it is so extensive that it is no localization at all, for antero-posteriorly and tranversely it includes the extreme capsule, the claustrum, the external capsule, the lenticular and caudate nucleus, the anterior and posterior segments of the internal capsule, and the optic thalamus.

This "quardilateral" corresponds in fact to the seat of predilection for softening of and haemorrhage into the brain, and it is incontestable, says Dejerine, that in the external lesions which are so often found at the autopsies of aphasics the "quadrilateral" is often, but not *constantly nor necessarily* injured,

whether in cases of motor, sensory, or total (Broca's) aphasia. Dejerine then proceeds to analyze with trenchant criticisms the anatomical connections and functions of the constituent parts of this "quadrilateral." The optic thalamus can be eliminated as no one has attributed to it the rôle of language.

As to the lenticular nucleus, to which Marie is inclined, to say the least, to attribute a language function, the study of secondary degenerations shows that the lenticular-caudate nuclei have practically no connection with the cortex, but that they are united with the optic thalamus and sub-thalamic regions, and *do not send any fibres* into the foot of the cerebral peduncles. To attribute to the lenticular nucleus and to its "afferent and efferent fibres a considerable part in phonation" is a pure hypothesis without basis in any known fact. It is interesting to know, particularly in view of recent papers supporting this hypothesis (Mills and Spiller, Dana), that Dejerine refuses to admit that this nucleus plays any part in the mechanism of phonation. If a lesion of this part accompanies dysarthria, the symptoms depend upon the concomitant injury of the internal capsule. As is well known, a unilateral lesion of the knee and anterior part of the posterior limit of the internal capsule produces pseudo-bulbar paralysis, from injury to the fibres coming from the cortical centres for the face, tongue, etc.

There remains then in the "quadrilateral" for examination, the internal capsule and the externo-extreme capsule. The internal capsule is composed of fibres whose origin and ending is known, and may be eliminated also. The constituent fibres of the externo-extreme capsule are then analyzed and discarded as positive factors in the problem, but considering the mode of arterial irrigation of this capsule, and its connection with the third frontal and the Rolandic operculum, it is not surprising that it is often found involved in the original lesion, or only degenerated when it escapes. We do not know a case where a lesion limited exclusively to the external capsule has produced pure motor aphasia, while on the contrary, cases of lesions of Broca's convolution occur when the external capsule is remarkably intact, *e g*, the two observations reported by Dejerine.

Recently Marie has discussed the superior and inferior limits of his "quadrilateral," basing his conclusions on the case

of MM. Laignel-Lavastine and Solomon. Dejerine, analyzing the anatomical findings in this case, finds that the lesion lay *above* the central ganglia, occupied the ascending parietal, penetrated into the centrum ovale, cut the white substance underlying the Rolandic operculum, and partly that of the *third frontal*. The centrum ovale being made up of conducting fibres alone is not in question, nor is the Rolandic region. The third frontal Marie rejects as a region of language. What then becomes, Dejerine asks, of the lenticular nucleus as playing a considerable rôle in the mechanism of language? Marie, he thinks, "is thus approaching more and more the *classical* location of pure motor aphasia called subcortical, for the lesion of the centrum ovale above the lenticular nucleus is that which was found in my (Dejerine's) original cases of motor subcortical aphasia (1891)". . . .

Dejerine then points out the different fibres passing in the centrum ovale *above* the lenticular nucleus and argues that, as it contains *the mass of fibres issuing from the region of Broca, if pure motor aphasia* ("anarthria") can result from a lesion of the white mass, it must be because of impairment of the function of this gray matter from which the white fibres issue. "There is no other interpretation possible unless we endow the white matter with functions which hitherto have been the property of gray matter."

Dejerine then analyzes a recent case reported by Marie and Montier in which there was "total — Broca's" aphasia, and in which a lesion involving Wernicke's zone and the *two Rolandic convolutions* on the left side, was found, on *macroscopical examination* (the brain was not sectioned). He argues that assuming that the macroscopic appearance showed the whole lesion, it could not have produced a motor aphasia, but only, as every one knows, a *paralysis*, and implies that the motor aphasia should be interpreted as one of involvement of the foot of the third frontal convolution which is inserted into the destroyed operculum.

There remains to consider the difference in the views of Marie and Dejerine regarding the nature of the intellectual defect, resulting from a lesion of Wernicke's zone, by which there is failure of comprehension of language. Marie makes much of his contention that this is a special intellectual defect, one of

intellectual elaboration, and involves the "*stock* of knowledge acquired by didactic procedures." In this article Dejerine does not discuss this question, but considers, as is well known, the failure of comprehension to be due to loss of sensorial images.

The criticism which one is entitled to pass upon these views is that so long as both (and the same is true of other writers) confine themselves to the most superficial clinical findings and the anatomical defects, neither is qualified to hold any opinion or pass any judgment upon it whatever. Marie boasts that he is a pure clinician and knows nothing of psychology in which he disclaims interest. He is satisfied to note a failure of comprehension of language as a whole, without further analysis and psychological investigation. He simply postulates a defect of "elaboration" involving "the stock of knowledge acquired by didactic procedures." We are entitled to ask, elaboration of what? Of memories of movements, or sounds, or visual images, or what?

The loss of comprehension of language is a pure psychological question, and should be studied as such, and until the defect is so studied it is more than naïve, it is childish, to postulate "intellectual defects" which tell us nothing. There are some psychological data which support the view that so-called word deafness is due to loss of the auditory "images" of words, but the problem deserves further and deeper investigation, and until this is done the less discussion about such "doctrines of aphasia" the better.

MORTON PRINCE.

THE MAJOR SYMPTOMS OF HYSTERIA. *By Pierre Janet. The Macmillan Co., New York and London. pp. vii + 345.*

These fifteen interesting lectures on hysteria, delivered during the season of 1906 at the Medical School of Harvard University, represent the latest French researches on the psychology of this protean disease, as given by one of its foremost students. In the first lecture the author states briefly the important data of the problem of hysteria and points out the necessity for a psychological study of the neuroses. The second and third lectures deal with fugues, and monoideic and polyideic somnambulisms. A number of interesting cases are cited and analyzed,

both from his own observation and from the reports of others. Such cases are typical forms of hysterical accidents, and are due to complete dissociations of consciousness, characterized by amnesia and an exaggerated and independent development of the emanicipated idea or ideas. Where one idea is dissociated he applies the term monoideic; where there is a dissociation of a system of ideas, the term polyideic is applied. When there is an aimless wandering and the somnambulistic episode is more or less protracted, it is called a fugue. The fourth lecture deals with the interesting subject of double personalities. The author points out how one state can dominate over the other and how in these cases of alternating personality the mental level rises and falls, thus producing the pluritic amnesia.

Lecture V. is taken up with the convulsive attacks, fits of sleep and the artificial somnambulisms. The inferior forms of somnambulism are hysterical fits and hysterical sleep, and these latter are frequently paralytic phenomena. The fits are imperfect forms of somnambulism, often nothing but aborted somnambulistic attacks. In hysterical sleep there is a decided lowering of all the sematic functions. All hysterical accidents can be artificially produced, especially the somnambulistic states. Hypnotism is artificial somnambulism, the hypnotic state has never any character which cannot be found in hysterical somnambulism, and the subjects are mostly hysterics. The author dogmatically asserts that it is impossible to hypnotize the epileptic or psychasthenic. Hypnotic states exactly resemble hysterical somnambulism, except that they are developed artificially instead of spontaneously.

To the motor phenomena of hysteria, the motor agitations, contractures and paralyses, two lectures are devoted (VI and VII). In all the motor disturbances of the limbs the mind is intact. These disturbances are divided into phenomena of exaggeration of movement, such as tics, choreas and contractures, and into phenomena of deficient motion or the paralyses. In differentiating between the psychasthenic and hysteric tic, Janet states that the former are accompanied by consciousness and attention and when the latter is not inhibited there is an increase of the tic. Distraction decreases the tic. In hysteria the opposite holds true, the movements are impeded by attention and

become more regular in distraction. Every type of tremor may be found in hysteria. Hysterical anaesthesia is a certain species of absent-mindedness, the sensation itself has not disappeared, but is merely dissociated, that is, not connected with the totality of consciousness. The same holds true of the hysterical amnesias; there is no real oblivion or destruction of memorial images, but merely an impossibility of the reproduction of these images. In hysterical paralysis, it is the idea of the motion of the limb that is lost and not the motion itself. Sometimes this loss comprises an entire system of images of movements, as in astasia-abasia.

Lecture IX is devoted to the hysterical disturbances of the special senses, particularly the disorders of vision. Here the contraction of the visual field, the blindness and the hemianopsia, are due to the same dissociation of the visual system as applies to the motor and sensory phenomena. There is no real blindness in hemianopsia, the visual stimuli are merely suppressed.

Lecture X is taken up with the speech disturbances. Janet discusses mutism, aphasia, aphonia, etc., shows their contradictory characteristics, and how absolutely unassociated they are with any organic changes either in the brain or vocal cords.

Lectures XI and XII are devoted to the disorders of alimentation and respiration. Much space is devoted to hysterical anorexia, which Janet believes is due to an anaesthesia of the stomach, to disturbances of organic sensibility with loss of hunger sensation. All types of respiratory tics may occur, peculiar barks, hiccoughs and almost coprotalic ejaculations. An hysterical Cheyne-States respiration is described, which possess the peculiarity that the respiratory rythm becomes normal when the patient is distracted.

In lecture XIII Janet discusses the stigmata of hysteria. After dismissing anaesthesia and falsehood as necessary accompaniment of hysteria, he states that the most fundamental stigma of hysteria is suggestion, and he again points out the marked unity between experimental suggestion (hypnosis) and spontaneous suggestion (hysteria), thus agreeing with the recent conceptions of Grasset. To the hysterical stigmata the fourteenth lecture is devoted. He divides these into two classes, the proper and common. The former occur only in hysteria, the latter are also found in the closely related neuroses, neurasthenia and psychasthenia.

Suggestion is the development of an idea, abstraction is exaggerated absent-mindedness, and both exist to an astonishing degree in hysteria. The subconscious phenomena are the results of this disposition to an exaggerated absent-mindedness. To all these he applies the phrase "retraction of the field of consciousness"; the mind is too narrow to take in a number of ideas. In anaesthesia, sensation escapes personal perception, in paralysis, movement, etc. The more common stigmata of hysteria are the feelings of incompleteness, the need of attracting attention, abulia, etc. All of this has now prepared the way for the admirable psychological summary which comprises lecture XV. Ideas are very important in hysterical accidents, they are powerful, dominating and act upon the body in an abnormal manner. The retraction of the field of consciousness either gives too much power to certain ideas or certain ideas may drop out of consciousness. Hence on one hand the exaggerated mobility and in the other the peculiar amnesias, anaesthesias and loss of motor control (paralyses). Hysteria therefore is a disease of personal synthesis, a form of mental depression characterized by the retraction of the field of personal consciousness and a tendency to the dissociation and emancipation of the systems of ideas and functions that constitute personality. Its starting point is a depression, an exhaustion of the higher functions of the brain. The dissociation seems to follow several laws — it reacts most powerfully on a function that was weak and disturbed, the most complicated functions disappear and the particular function is inhibited that was in full activity at the time of the emotion.

We have given a very brief summary of a most important series of lectures. The stress laid upon the purely mental side of hysteria, as distinct from the classical physical symptoms, is a step in the right direction. These lectures of Dr. Janet form a pertinent example of the important results that can be attained by purely psychological studies.

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